AMENDMENTS TO THE SPECIFICATION:

Please amend the specification as shown:

Please amend paragraph [0091] beginning on page 35 as follows:

[0091] A fusion polypeptide of the disclosure can be produced by expression of polynucleotide encoding a fusion polypeptide in prokaryotes. These include, but are not limited to, microorganisms, such as bacteria transformed with recombinant bacteriophage DNA, plasmid DNA, or cosmid DNA expression vectors encoding a fusion polypeptide of the disclosure. The constructs can be expressed in E. coli in large scale for *in vitro* assays. Purification from bacteria is simplified when the sequences include tags for one-step purification by nickel-chelate chromatography. Thus, a polynucleotide encoding a fusion polypeptide can also comprise a tag to simplify isolation of the fusion polypeptide. For example, a polyhistidine tag of, *e.g.*, six histidine residues, can be incorporated at the amino terminal end of the fusion polypeptide. The polyhistidine tag allows convenient isolation of the protein in a single step by nickel-chelate chromatography. A fusion polypeptide of the disclosure can also be engineered to contain a cleavage site to aid in protein recovery or other linker moiety separating a PTD from a heterologous molecule. Typically a linker will be a peptide linker moiety. The length of the linker moiety is chosen to optimize the biological activity of the polypeptide comprising PTD domain and a heterologous molecule and can be determined empirically without undue experimentation. The linker moiety should be long enough and flexible enough to allow a PTD polypeptide to freely interact. A linker moiety is a peptide between about one and 30 amino acid residues in length, typically between about two and 15 amino acid residues. Examples of linker moieties are --Gly--Gly--, GGGGS (SEQ ID NO:4), (GGGGS)~~N~~ **XAA** (SEQ ID NO:5), GKSSGSGSESKS (SEQ ID NO:6), GSTSGSGKSSEGKG (SEQ ID NO:7), GSTSGSGKSSEGSGSTKG (SEQ ID NO:8), GSTSGSGKPGSGEGSTKG (SEQ ID NO:9), or EGKSSGSGSESKEF (SEQ ID NO:10). Linking moieties are described, for example, in Huston *et al.*, Proc. Nat'l Acad. Sci 85:5879, 1988; Whitlow *et al.*, Protein Engineering 6:989, 1993; and Newton *et al.*, Biochemistry 35:545, 1996. Other suitable peptide linkers are those described in U.S. Pat. Nos. 4,751,180 and 4,935,233, which are hereby incorporated by reference. A DNA sequence encoding a desired peptide linker can be inserted between, and in the same reading frame as, a

polynucleotide encoding a PTD polypeptide or fragment thereof followed by a heterologous polypeptide, using any suitable conventional technique. For example, a chemically synthesized oligonucleotide encoding the linker can be ligated between two coding polynucleotides. In particular embodiments, a fusion polypeptide comprises from two to four separate domains (*e.g.*, a PTD domain and a heterologous polypeptide domain) are separated by peptide linkers.

Please amend paragraph [00112] on page 45 as follows:

[00112] Peptide synthesis. All peptides (HA2-Tat: GLFGAIAGFIENGWEGMIDGGRKKRRQRRR (SEQ ID NO:11); Tat: GRKKRRQRRR) (portion of SEQ ID NO:11) were synthesized as D-amino acid, retro-inverso forms using solid-phase FMOC chemistry on an Applied Biosystems 431A synthesizer. Peptides were cleaved in 92.5% TFA, 2.5% H_2O, 2.5%thioanisole, 2.5 EDT for 5h hours, precipitated in ether and purified on C18 reverse phase HPLC column. Major peaks were analyzed by electrospray mass spectrography. Fractions corresponding to the correct molecular weight were lyophilized and stored at -80°C. Prior to use peptides were resuspended in PBS and sterile filtered. The concentration of peptide solutions was determined by absorbance at 215 and 225nM.

Please amend paragraph [00124] beginning on page 51 as follows:

[00124] Several viruses have evolved endosomal escape mechanisms to enter the cytoplasm by taking advantage of the vesicle low pH to induce protein conformational changes that trigger endosomal membrane destabilization 24. The N-terminal 20 amino acids of the influenza virus hemagglutinin (HA) protein, termed HA-2 (GLFGAIAGFIENGWEGMIDG) (SEQ ID NO:12), is a well characterized fusogenic peptide that has been shown to destabilize membranes at low pH. To increase the efficiency of TAT-fusion polypeptide release from macropinosomes, a proteolytically-stable, retro-inverso D-amino acid peptide corresponding to the HA-2 domain peptide followed by the TAT transduction domain (HA2-TAT) was synthesized. Treatment of tex.loxP.EG T cells with a sub-threshold concentration of TAT-Cre protein resulted in minimal recombination and expression of eGFP (Fig. 5b). In contrast, treatment of cells with TAT-Cre and in combination with HA2-TAT peptide resulted in

marked increases (>10-fold) in recombination. This enhanced effect appears unrelated to the TAT domain, as cells treated with control TAT D-isomer peptide showed only minor increases in recombination (Fig. 5c). Consistent with the lipid raft-dependent results above, pretreatment with nystatin inhibited HA2-TAT-mediated enhancement of recombination by TAT-Cre (Fig. 5d). Taken together, these observations demonstrate the ability to further enhance TAT-mediated transduction into the cells.